

1401 Village Blvd, Apt 1913
West Palm Beach, FL 33409
May 5, 2005

Ms. Cynthia Oshita
Office of Environmental Health
Hazard Assessment
Proposition 65 Implementation
Program
P.O. Box 4010
Sacramento, CA 95812-4010

Dear Ms. Oshita,

Please accept the following statement for
use with the meeting of May 9, 2005
for discussion of danger of acrylamide.

Sincerely
Richard Segal

OFFICE OF ENVIRONMENTAL HEALTH
HAZARD ASSESSMENT
Received

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SACRAMENTO

mutations found from glycidamide exposure² appear to suggest that carcinogenesis occurs under this condition. The genes in question include the HRR gene which seem to be site of deamination but glycidamide causes. There is a mouse mutation at the la gene which correlates to ethylene oxide carcinogenesis and the kind in use is from acrylamide study of 1986 B6C3F₁. The APC mutation occurs it is found with colon cancer and is related to diet. The P53 gene is mutated and this can cause numerous carcinogenesis phenomena based on failure of tumor suppression.

There would seem to be a repair failure of the polymerases use given the presence of mutations hereby cytosine is likely to be poorly repaired part of deamination DNA to uracil.

Some mutation leads to tumor growth
of prevention are directed to glutathione. The
glutathione useful for prevention of adducts is
not found as such with glycidamide exposure
possibly its control gene is mutated by glycidamide.

The question of the kinds of adducts in
question began with carboxyethyl, in vitro,
from acrylamide, followed with 2-carbamoyl-2-
hydroxyethyl from conversion to glycidamide
in vivo and has led to glycidamide

caused wudene adducts in vitro. Thus the
co-existence of adducts of danger are
known, and it would appear likely that
an adduct of cytosine deaminated to wudene
will mutate a human gene carcinogenesis

The repair failures noted of glycidamide
cause may turn out to be as well-understood
as those from benzo[a]pyrene, given work on
nucleotide excision repair and base excision
repair, repair units of polymerases and
glycosylases. Certainly with some of the

same genes mutated, similar repair failures be found. The cyclic adduct, postulated as part of the deamination in question, has repair deficiencies. In addition, its formation at cytidine is of scientific inquiry, given acrolein results

It is interesting that mutations of the *Drosophila* and *uracil*, shown from exposure to ethylene oxide and propylene oxide, the latter of which is known to deaminate in vivo, cannot be ruled out as the cause of nucleotide excision repair fault, when in comparison to guanine adducts

Uridine glycol part of mutation in vivo with
aracil glycol found to be un-repaired at
a low dose in the study of glycidamide, its
low-dose high-exposure carcinogenic factor
may be partially explained.

It is for these reasons that I agree that
a warning, such as the one devised which begins
"Baking, roasting, frying," should be provided to
consumers. I do not agree that an exemption
should be offered for acrylamide products. The
onset-of-cancer rate is one-in-three and is
approaching one-in-two. Cancer from smoking
declined while cancer from diet increased
acrylamide likely leads in carcinogenesis causes
among food products. Since the same Swedish
researchers have contributed to their epidemiology
with mutation studies which have found
agreement and advised mutational study
from other scientists their definition of risk
from acrylamide continues to be important
that 50 micrograms of acrylamide per day
can lead to in 0 cancer onset risk

The Non-significant Risk Level using this rate should be < 1.0 micrograms per day because of an occurrence of very low dose and high doses.

Furthermore, attention should be directed to at risk populations due to genetics. Given that the U.S. EPA was to be consulted rather than action taken as a result of the October 2003 Carcinogenic Identification Committee meeting it should be noted that leading acrylamide viewpoint at U.S. EPA includes criticism of the 2004 National Toxicology Program advisory as being too weak regarding developmental effects (Dearfield). This view is in agreement with Swedish viewpoint (Abrahamsson Zetterberg) which extends the risk to 1 in 100 for 130 micrograms of acrylamide per day.